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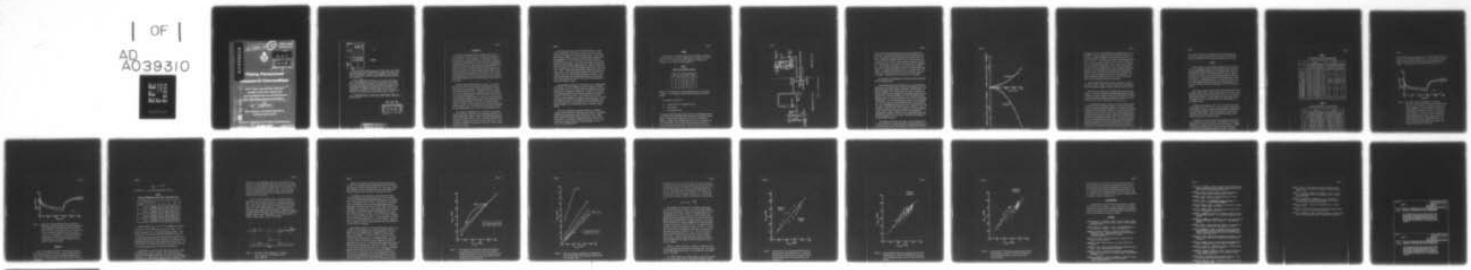

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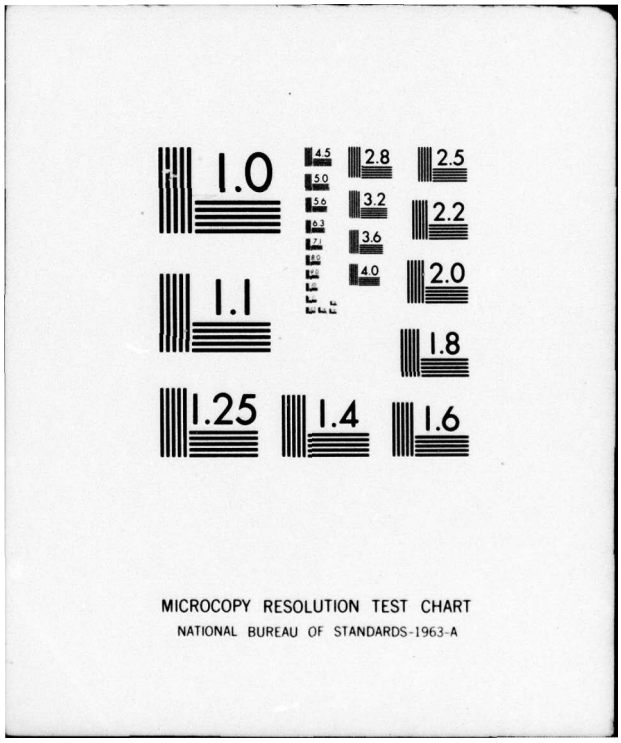
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Flying Personnel Research Committee

(6) End Tidal and Mixed Venous Carbon Dioxide Tensions During Exposure to and Recovery from Voluntary Hyperventilation,

by

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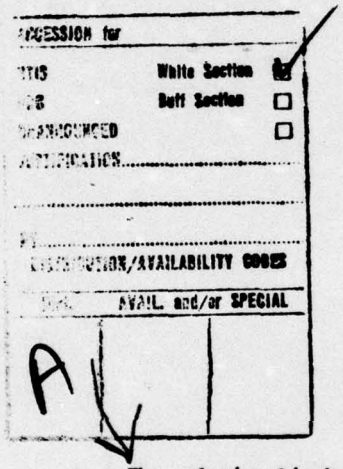

ABSTRACT

The relationship between end tidal and mixed venous carbon dioxide tensions has been studied during exposure to, and recovery from, voluntary hyperventilation in 4 subjects. The experiments were designed to study the feasibility of using a single rebreathing estimate of mixed venous carbon dioxide tension as a simple field test for hyperventilation in pilots.

The results confirmed that the fall of end tidal carbon dioxide tension during hyperventilation and rise during recovery was exponential. The results also showed that the relationships between mixed venous and end tidal carbon dioxide values during the unsteady states of carbon dioxide washout and accumulation may be described as a loop which encloses the theoretically derived line for the steady state relationships. The deviation from the steady state line appears on theoretical consideration to be directly proportional to carbon dioxide elimination rate, and indirectly proportional to cardiac output.

It is concluded that a field test for hyperventilation based on a single rebreathing estimate of mixed venous carbon dioxide tension would not be of value.

INTRODUCTION

The effects of hyperventilation on cerebral function are of obvious importance in aviation, where a decrement in performance can lead to disaster. In 1941, Rushmer et al, and Hinshaw & Boothby stressed that even mild hyperventilation could produce a degradation in performance, and Hinshaw et al, (1943) stated that prolonged mild hyperventilation could be as deleterious as profound hyperventilation of shorter duration. That hyperventilation was associated with a reduction in cerebral blood flow was shown by Kety & Schmidt (1946), and they ascribed this to cerebral vasoconstriction. Meyer & Gotoh (1960) showed that the appearance of delta wave activity on the EEG during hyperventilation was the same as that seen in cerebral hypoxia. Further physiological effects of overbreathing have been reviewed by Brown (1953).

The major cause of hyperventilation is anxiety. In aviation, there is little doubt that the wearing of oxygen apparatus with an added resistance to breathing can cause hyperventilation in susceptible subjects (Ernsting, 1965). In addition, Miles (1957) showed that 15 of 32 experienced divers overbreathed at rest when attached to a standard diving mouthpiece. Other factors that could play a part in the aetiology of hyperventilation in the air have been identified, such as pressure breathing (Ernsting, 1964), chest compression by seat restraint harnesses (McIlroy et al, 1962), certain body movements (Dixon et al, 1962), vibration (Ernsting, 1961), and raised body temperature (Saxton, 1975). The additive effect of positive acceleration has been identified (Brent et al, 1957) and studied (Browne, 1959).

The incidence of hyperventilation in flight has been studied in the past in two ways. The first is the retrospective analysis of in-flight incidents and accidents. Konecci (1956) identified hyperventilation as the causative factor in 8 of 73 in-flight incidents in the T33 aircraft. Powell et al, (1957) described 3 causes of loss of consciousness or diminished consciousness in pilots as a result of hyperventilation in flight.

The second approach is to study in-flight ventilation. Norris (1964) concluded that respiratory rate alone did not provide enough evidence for a diagnosis of in-flight hyperventilation. Ellis & Wells (1962) produced evidence to show that the measurement of several acid-base components in blood and urine could detect previous in-flight hyperventilation 'at a level of concern' but that lower levels of hyperventilation could be missed. Balke (1956, 1957) studied in-flight ventilation, and concluded that hyperventilation in flight was common, and that the incidence rose with the performance capabilities of the aircraft flown. Murphy & Young (1968) measured minute volumes during flights in light aircraft. They found increased ventilation during take-off, and a smaller increase in ventilation during approach and landing. They also found that amateur pilots hyperventilated more than professional pilots, and that the first flight of the day elicited more hyperventilation than subsequent flights.

The retrospective technique has the weakness that it underestimates the incidence. Some cases (especially second or subsequent episodes) are not reported, as the pilot finds himself discouraged by his previous experience of the subsequent investigation. Secondly, it is difficult to distinguish hyperventilation arising from hypoxia from hyperventilation due to other causes; finally, the incidents that end in fatal crashes are almost impossible to diagnose as hyperventilation. The study of in-flight ventilation has the drawbacks that the experimental technique often interferes with the primary task of flying, and also that present equipment is not electronically stable at altitude or small enough.

The series of experiments reported here began as an attempt to develop a simple field test for hyperventilation, based on a rebreathing technique for measuring mixed venous carbon dioxide levels ($P_{\bar{v}}CO_2$). Before immediate post-flight rebreathing estimates of $P_{\bar{v}}CO_2$ could be entertained, it was necessary to define what happened to the end tidal carbon dioxide ($P_{ET}CO_2$) and $P_{\bar{v}}CO_2$ values during exposure to, and recovery from, hyperventilation.

METHODS

Four subjects, 3 of whom were experienced in respiratory manoeuvres, were used throughout the series of experiments, and details of them are shown in Table 1. Before starting the experiment each subject was thoroughly trained in the use of the apparatus.

TABLE 1

Details of Experimental Subjects

Subject	Age	Height cm	Weight kg
MG	27	181.2	78.5
JG	29	176.9	66.5
BP	35	182.2	77.5
CS	32	172.2	69.5

Subjects lay horizontally on a couch, breathing from the circuit shown in Figure 1. The apparatus was essentially that described by Denison (1969).

The apparatus consisted of:

a. A breathing circuit for hyperventilation.

b. A gas analyser.

c. A rebreathing circuit.

The hyperventilation breathing circuit consisted of compressed gas sources controlled by pressure reducing valves and monitored by accurate pressure gauges. The gases passed through sonic orifices to a mixing flute, through a rotameter and low pressure wide bore hosing to a reservoir bag. The largest bulk gas (air) entered the mixing flute most distally in order to wash the other gases along. From the reservoir bag, the gas

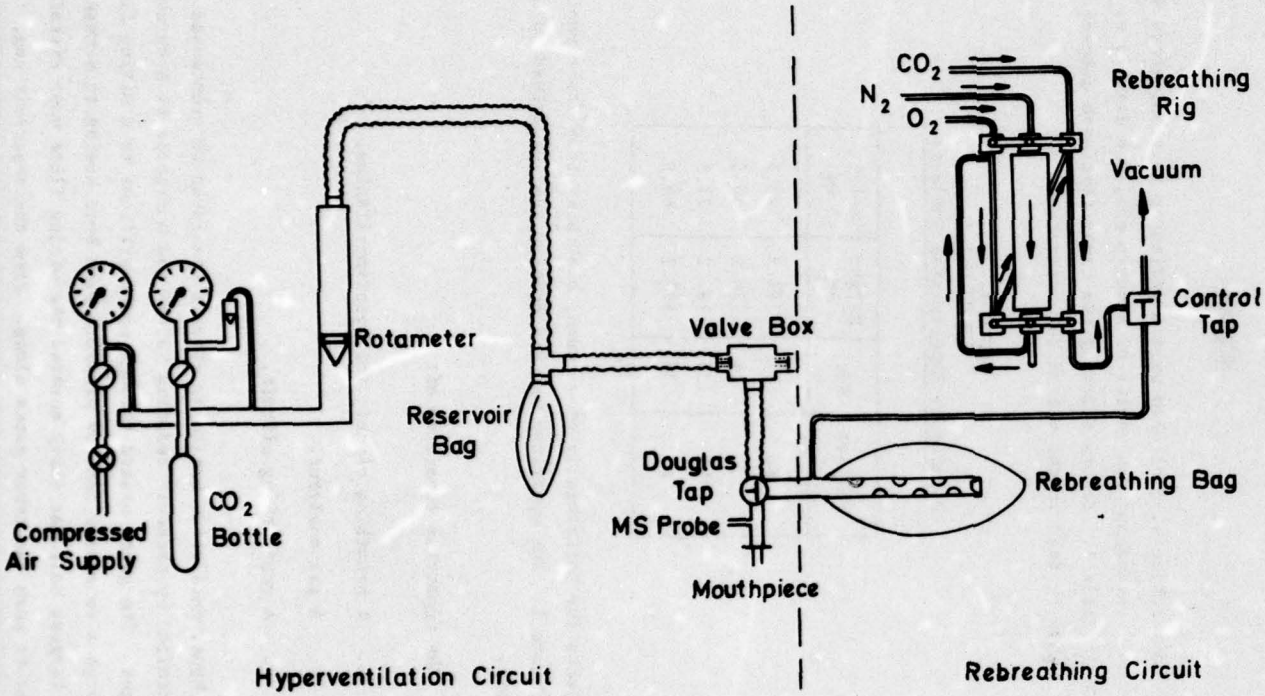

Figure 1. Breathing Circuit.

passed through a spring loaded non-return valve and a Douglas tap to the subject. Expiration was through a second spring loaded non-return valve. The reservoir bag was large enough (5L) and the valves so set that no through flow to ambient occurred until the subject had missed 3 breaths at a $\dot{V}_I$ of 30 L/min. Minute volume was monitored on a rotameter (10-100 L/min), and altered by controlling the flow of compressed air. Carbon dioxide could be added to the inspirate in the mixing flute, and its flow was separately monitored on a rotameter (100-1,000 ml/min). An automatic timer/bleeper gave the inspiratory signal to the subject; at each bleep he had to inspire from the reservoir bag until it was empty. Respiratory rate was controlled at 20 breaths/min.

The resistance to flow presented by the hyperventilation breathing circuit is given in Fig 2.

The rebreathing circuit consisted of the apparatus to provide the gas mixtures, a 5L rubber bag and a Douglas tap. Compressed gas bottles supplied N_2 labelled with 5% Argon, O_2 and CO_2 to three reservoirs, of which the reservoir for the inert gas was the largest. The amount of each gas delivered could be changed by altering the appropriate pressure reducing valve. The entry and exit taps of the three reservoirs were ganged in parallel so that the reservoirs could be filled and discharged simultaneously. The circuit was arranged so that the inert gas flushed the other two reservoirs as the gas was discharged to the rebreathing bag. The rebreathing bag could be evacuated by a vacuum pump after each rebreathing. The subject was instructed, after a countdown from 5 to 0, to exhale to residual volume, turn the Douglas tap into the rebreathing circuit, inhale to empty the bag, and then to breathe maximally at approximately 1 cycle/sec to achieve as much gas movement between lungs and bag as possible until told to stop. The subject then returned to the hyperventilation circuit.

Two pulmonary ventilations were studied - minute volumes ($\dot{V}_I$) of 20 L/min and 30 L/min. Subjects started the experiment breathing at one or the other minute volume, with CO_2 added to the inspirate to maintain

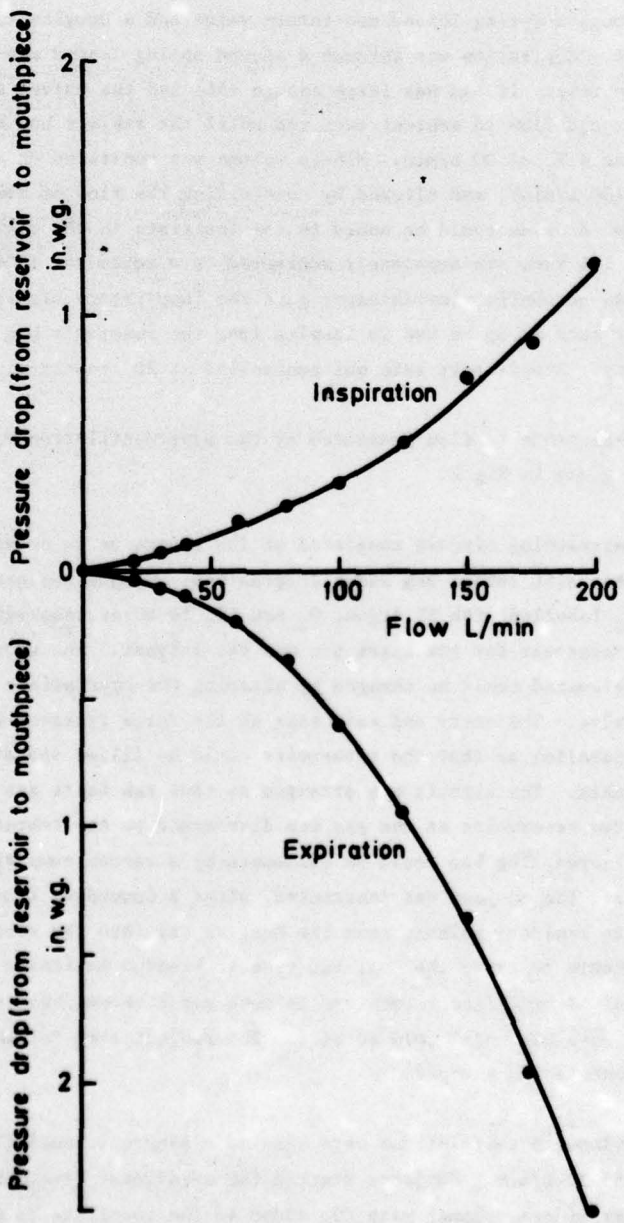

Figure 2. Pressure/Flow Characteristics of Hyperventilation Circuit.

the $P_{ET}CO_2$ at 38.5 Hg. The experiments were performed with the $\dot{V}_I$ set throughout, in order to maintain the work of breathing constant. In addition, the $P_{ET}CO_2$ starting point was controlled to attempt a more satisfactory control over $P_{\bar{v}}CO_2$, thus allowing a standard platform from which to launch into the CO_2 washout. After two control estimates of $P_{\bar{v}}CO_2$ had stabilised again at 38.5 mm Hg, the added CO_2 was withdrawn from the inspirate, allowing washout of CO_2. After the washout had continued for the period under study, a further estimate of $P_{\bar{v}}CO_2$ was obtained. CO_2 was then added to the inspirate at the same rate as during the initial control period, while the subject maintained the same $\dot{V}_I$. $P_{\bar{v}}CO_2$ was measured at set intervals during recovery. The periods of washout studied were ½ min, 1, 2, 5, 10, 30 and 60 min. During this time, $P_{ET}CO_2$ was monitored continuously.

As Farhi & Rahn (1960) pointed out, the body stores of CO_2 can take several hours to adjust. With this in mind, no subject performed more than one washout of greater duration than 10 min in any one day.

Monitoring of the composition of respired gases was by mass spectrometry. An AEI MS4 and then an SRI MS8 mass spectrometer were used, and these were linked to direct writing, hot stylus recorders. Calibration gases, previously analysed by Lloyd-Haldane apparatus, were used which spanned the range of expected values of P_{O_2} and P_{CO_2}.

In a rebreathing estimate of $P_{\bar{v}}CO_2$, a gas-filled, bag-lung system is allowed to mix, and the gases equilibrate. The early part of the record is distorted by mixing, the middle part is a plateau whose level is determined by the volume and composition of the gas in the bag and the $P_{\bar{v}}CO_2$, and the latter part of the record can be distorted by recirculation, and intersolubility effects. Mixing of the lung-bag system was monitored by observing the equilibration of the argon in the nitrogen as it cycled between bag and lung. $P_{\bar{v}}CO_2$ values were obtained by measuring the plateau level of CO_2 achieved between the 6th and 10th seconds of rebreathing. On the few occasions when a satisfactory equilibrium (i.e. simultaneous plateaux on the O_2 and CO_2 traces, each

with a slope of less than 0.1 mm Hg/sec) was not achieved, $P_{\bar{v}}CO_2$ was obtained in the manner described by Denison (1969), by extrapolating the 6 to 10 second line to 20 seconds, and taking that as the value.

RESULTS

Subjects reported the usual symptoms during the period of hypocapnia, i.e. paraesthesiae, muscular spasms, sweatiness, cold extremities, sleepiness and diminished awareness. It was noted, however, that subjects became less aware of the paraesthesiae after 30 min of hyperventilation at 30 L/min and that the muscular spasms appeared to remit at the same time. It is not known whether this was a physiological effect or the effect of diminished awareness. It is worth noting that no subject in the present study became unconscious, although $P_{ET}CO_2$ fell to as low as 9.7 mm Hg in one subject and $P_{\bar{v}}CO_2$ to as low as 12.1 mm Hg in another.

Mean values of $P_{ET}CO_2$ during 20 L/min and 30 L/min washout/recovery are shown in Tables 2 and 3. Mean values of $P_{\bar{v}}CO_2$ during 20 and 30 L/min washout and recovery are also shown in Tables 2 and 3. Full data may be obtained from the author. The mean washout and recovery $P_{ET}CO_2$ values are graphed in Fig 3, and the mean washout and recovery $P_{\bar{v}}CO_2$ values are graphed in Fig 4.

The mean control $P_{ET}CO_2$ was 38.3 ± 0.9 (s.d.) mm Hg, and the mean control $P_{\bar{v}}CO_2$ was 45.6 ± 1.7 mm Hg. Control of $P_{ET}CO_2$ was satisfactory and there was no significant difference between control values obtained at the two minute volumes. Control of $P_{ET}CO_2$ then controlled $P_{\bar{v}}CO_2$ adequately, and there was no significant difference between control values obtained at the two minute volumes.

Once washout of CO_2 started, there was an immediate difference in $P_{ET}CO_2$ between the two minute volumes. The difference was very highly significant ($P < 0.001$) even after only 10 seconds of washout. Washout of CO_2 during hyperventilation at 30 L/min was more severe than at 20 L/min at all times during the washout ($P < 0.001$). During the

TABLE 2

Mean Washout Values of $P_{ET}CO_2$ and $P_v\bar{C}O_2$

Time	$P_{ET}CO_2$					$P_v\bar{C}O_2$				
		20 L/min		30 L/min			20 L/min		30 L/min	
	n	mm Hg	se	mm Hg	se	n	mm Hg	se	mm Hg	se
0	56	38.2	0.1	38.4	0.1	56	46.0	0.2	45.3	0.2
10 sec	28	33.0	0.3	30.4	0.2	-	-	-	-	-
20 sec	28	31.5	0.3	28.8	0.3	-	-	-	-	-
30 sec	28	30.6	0.3	27.9	0.3	4	43.6	0.6	41.4	0.3
40 sec	24	30.0	0.4	27.2	0.3	-	-	-	-	-
50 sec	24	29.5	0.3	26.7	0.3	-	-	-	-	-
1 min	24	29.2	0.3	26.0	0.3	4	41.0	1.0	39.4	0.2
2 min	20	27.3	0.4	23.6	0.3	4	37.8	0.6	35.2	0.7
5 min	16	24.2	0.4	20.3	0.2	4	33.4	0.6	31.3	0.6
10 min	12	22.5	0.5	17.2	0.5	4	29.1	0.6	24.6	0.9
15 min	8	21.3	0.6	16.0	0.5	-	-	-	-	-
20 min	8	19.8	0.7	15.2	0.5	-	-	-	-	-
25 min	8	18.6	0.7	14.5	0.6	-	-	-	-	-
30 min	8	18.0	0.7	13.8	0.6	4	26.3	0.5	19.9	1.5
35 min	4	17.1	0.9	13.3	0.9	-	-	-	-	-
40 min	4	16.4	0.8	12.7	1.2	-	-	-	-	-
45 min	4	15.9	0.9	12.4	1.3	-	-	-	-	-
50 min	4	15.7	0.8	12.2	1.4	-	-	-	-	-
55 min	4	15.2	0.7	12.0	1.3	-	-	-	-	-
60 min	4	14.9	0.8	11.8	1.2	4	19.8	0.9	15.5	1.5

TABLE 3

Mean Recovery Values of $P_{ET}CO_2$ and $P_v\bar{C}O_2$

Time min	$P_{ET}CO_2$				$P_v\bar{C}O_2$			
	20 L/min		30 L/min		20 L/min		30 L/min	
	mm Hg	se	mm Hg	se	mm Hg	se	mm Hg	se
2	23.7	0.6	25.4	0.9	25.9	1.1	23.8	1.0
5	27.9	0.8	30.6	0.9	29.7	1.1	29.6	1.0
10	30.4	0.8	34.4	0.9	34.0	1.0	35.8	1.3
15	31.7	0.8	36.0	0.5	36.8	1.7	39.6	0.6
20	32.7	0.7	36.5	0.2	38.7	1.3	41.6	1.0
25	33.6	0.8	37.0	0.3	40.3	1.5	43.3	1.3
30	34.4	0.7	37.5	0.4	40.7	0.8	44.1	1.1
35	35.4	0.7	37.6	0.4	40.8	1.0	44.1	1.3

recovery, however, $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ at a $\dot{V}_I$ of 30 L/min recovered faster than at a $\dot{V}_I$ of 20 L/min (see Figs 3 and 4). This is because the experimental design would make the recovery tend to be a reciprocal of the washout. The statistical analyses used were analyses of variance.

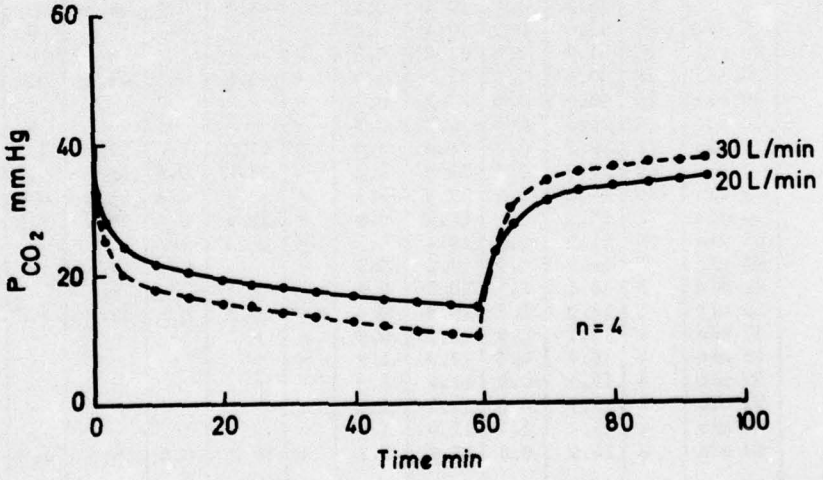

Figure 3. Mean Values of $P_{ET}CO_2$ During Washout and Recovery. The values from the washout are from all experiments; mean values of $P_{ET}CO_2$ in the first 30 seconds of washout are *therefore* means of 28 values; similarly, means at 35 min and subsequently are means of 4 values. The recovery values are means of recovery from 60 min of washout and are therefore means of 4 values. It is not possible to show the standard errors on this Figure; 46 of the 56 points depicted would have standard error bars of less than ±1.0 mm long on this scale, and the greatest standard error bar would be ±1.8 mm long.

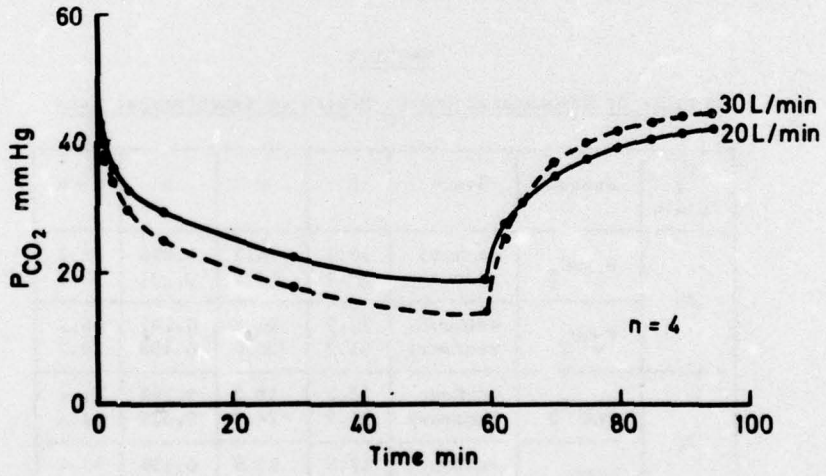

Figure 4. Mean Values of $P_v\text{-}CO_2$ During CO_2 Washout and Recovery. Each $P_v\text{-}CO_2$ obtained during washout represents the endpoint of one experiment, and the mean $P_v\text{-}CO_2$ is therefore the mean of 4 values. The recovery means are of recovery from 60 min of washout and the means are therefore means of 4 values. It is not possible to show the standard errors on this Figure; 6 of the 16 points depicted would have standard error bars of less than ±1.0 mm long, and the greatest would be ±1.9 mm long.

DISCUSSION

Since, as was expected, the shape of the washout and recovery curves appeared to follow a power law, a computer program was used to try to fit exponentials to the values of $P_{ET}CO_2$ and $P_v\text{-}CO_2$ given in Tables 2 and 3. Single exponential curves were fitted in the form of the equation

$$P_{CO_2} = c + b.e^{-at} .$$

The values of c, b and a obtained are shown in Table 4.

TABLE 4

Details of Exponential Curves Fitted to Experimental Data

$\dot{V}_I$ L/min	Measure	State	c	b	a	c + b
20	$P_{ET}CO_2$	washout	16.1	16.1	0.094	32.2
		recovery	33.7	-17.9	0.221	15.8
	$P_{\bar{v}}CO_2$	washout	22.9	21.8	0.141	44.7
		recovery	41.3	-20.6	0.108	20.7
30	$P_{ET}CO_2$	washout	13.2	18.2	0.269	31.4
		recovery	36.7	-24.3	0.316	12.4
	$P_{\bar{v}}CO_2$	washout	17.5	25.9	0.136	43.4
		recovery	44.3	-27.9	0.125	16.4

From the equation $P_{CO_2} = c + b.e^{-at}$, the asymptote is given by the term (c). The intercept on the P_{CO_2} axis (i.e. when t = 0) is given by the term (c + b). These values are shown in Table 4. Thus the P_{CO_2} value at the start of hyperventilation washout is given by (c + b) and the value to which the washout proceeds is given by (c); the value at which recovery starts is given by (c + b) from the recovery exponentials and the level at which recovery ends is given by (c). It can be seen that the estimated intercepts on the P_{CO_2} axis at time zero for the $P_{ET}CO_2$ washout states are lower than those observed.

Many studies, e.g. Farhi and Rahn (1955), Vance and Fowler (1960) and Stoddart (1965) accept a 'body pool' idea for CO_2 stores; the latter two studies suggest that washout occurs from a multicompartment body store. It is clear therefore that more than one exponential washout of CO_2 is seen at the lips during hyperventilation. These workers

demonstrate a 'lung' compartment of CO_2 with a very short half life. The loss of the lung compartment washout data in the first few seconds of washout could explain why the single exponential fitted curves start lower on the P_{CO_2} axis than the observed curves. This is not seen in the $P_{\bar{v}}CO_2$ curves because the estimation of $P_{\bar{v}}CO_2$ by the rebreathing method necessitates equilibration of the venous blood and lung compartments with the bag of gas.

It may also be seen from Table 4 that the washout and recovery exponentials, apart from being inversions of each other, differ in shape. This could be because recovery was interspersed with rebreathings. Figure 5 shows $P_{ET}CO_2$ during recovery firstly less than P_ICO_2 (section A), equal to P_ICO_2 (section B), a rebreathing (section C) and finally $P_{ET}CO_2$ greater than P_ICO_2 (section D). Simple calculation can demonstrate that when $P_{ET}CO_2$ < inspired PCO_2 (P_ICO_2) the rebreathing manoeuvre hinders recovery, when $P_{ET}CO_2 = P_ICO_2$ it has no effect on recovery, and when $P_{ET}CO_2 > P_ICO_2$ it aids recovery.

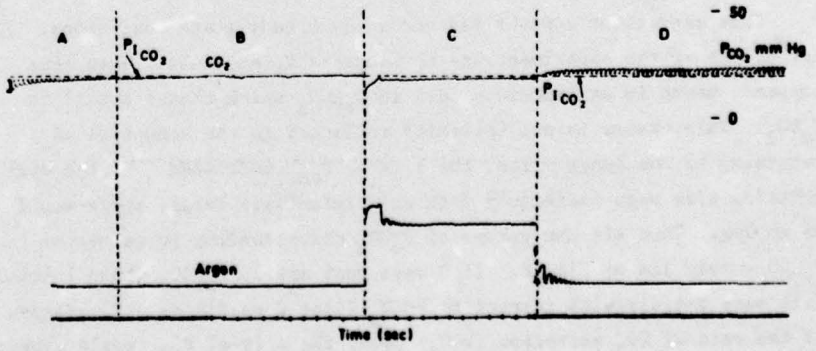

Figure 5. Experimental Trace, showing $P_ICO_2 > P_{ET}CO_2$ (A) $P_ICO_2 = P_{ET}CO_2$ (B), a rebreathing (C), and $P_ICO_2 < P_{ET}CO_2$ (D).

Because we are interested in detecting previous hyperventilation in a pilot, a rebreathing estimate of $P_{\bar{v}}CO_2$ would have to be performed very soon after the end of hyperventilation to be of any value. Furthermore, diagnosis of previous hyperventilation by a single rebreathing estimate of $P_{\bar{v}}CO_2$ would be unreliable, for reasons that follow.

If a straight line CO_2 dissociation curve existed, the steady state relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ would be a line parallel to the line of identity passing through the point ($P_{\bar{v}}CO_2$ = 45.6, $P_{ET}CO_2$ = 38.3). This appears as line A in Fig 6. The effect of the alinearity of the CO_2 dissociation curve on the relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ may be calculated from the data of Kelman (1967). The resulting line appears as line B on Fig 6. This line was calculated for an oxygen uptake ($\dot{V}_{O_2}$) of 250 ml/min, a Respiratory Exchange Ratio (R) of 0.85, and a cardiac output ($\dot{Q}$) of 5 L/min. In the steady state, a change of $\dot{V}O_2$ or R or $\dot{Q}$ will change the slope of the line. The effect of changing R was calculated and is shown in Fig 7. It can be seen that as R tends to zero, so the relationship tends to the line of identity. Similarly, when R is negative (e.g. during CO_2 assimilation) the relationship would lie below the line of identity.

This experiment however did not study steady state conditions. The net effect of the experiment was to increase $\dot{V}_A$ suddenly. When this happens, there is an immediate fall in $P_{ET}CO_2$ which causes a fall in P_aCO_2. This change is not initially reflected in the venous blood returning to the lungs - i.e. the Δ (v-a) P_{CO_2} increases. If the circulation time were infinitely fast or $\dot{Q}$ infinitely large, there would be no lag. Thus all the values of $P_{\bar{v}}CO_2$ corresponding to values of $P_{ET}CO_2$ would lie on line B. If $\dot{Q}$ were very small, $P_{ET}CO_2$ would initially fall very rapidly with respect to $P_{\bar{v}}CO_2$ (line C on Fig 6). Similarly, if the rate of CO_2 excretion ($\dot{V}CO_2$) rose, the Δ (v-a) P_{CO_2} would appear greater, and the initial deviation from line B would be greater. If $\dot{V}CO_2$ fell until a steady state existed, the deviations from line B would be zero. During recovery, the reverse arguments may be applied, and the relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ would lie on the other side of the steady state line. Again the deviation would be affected

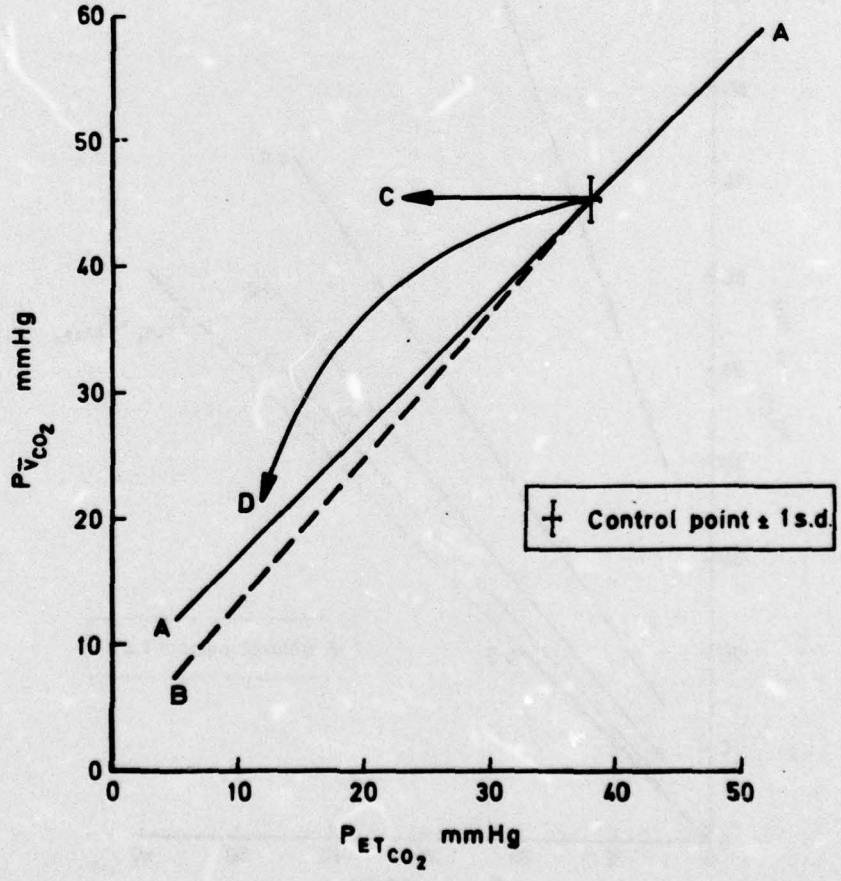

Figure 6. The "Steady State" Relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ during Hyperventilation-Theory, where line A assumes a straight CO_2 dissociation curve, line B assumes the alinear CO_2 dissociation curve, and lines C and D show the effect of departure from the steady state.

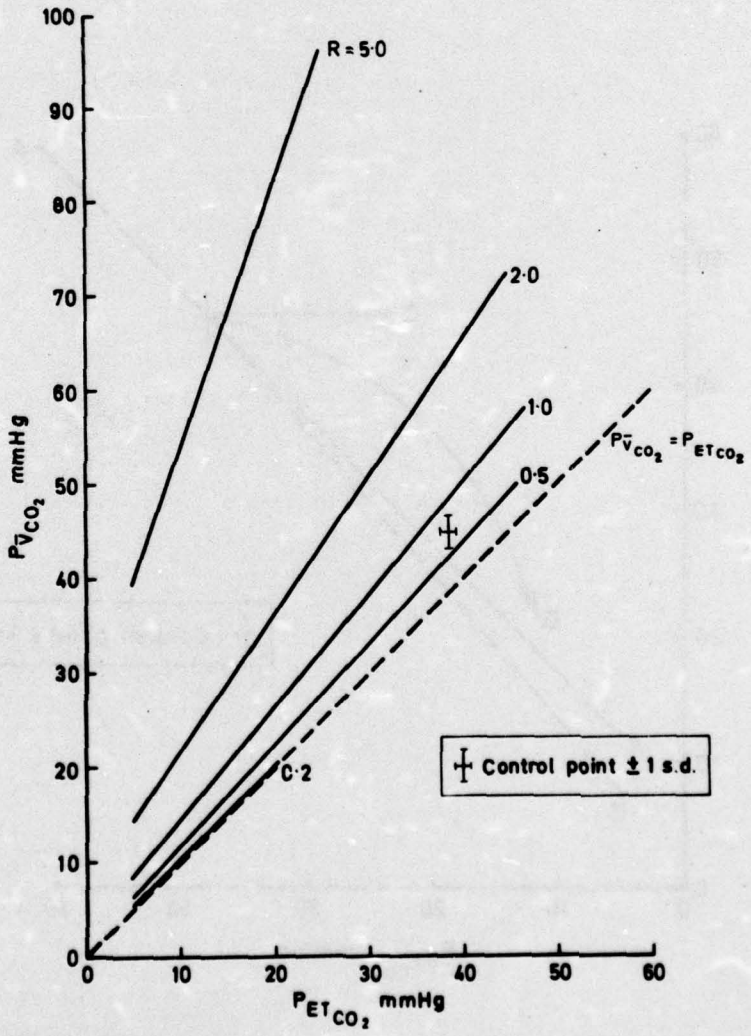

Figure 7. Effect of Respiratory Exchange Ratio on Steady State Relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ (calculated from data of Kelman, 1967).

by $\dot{V}CO_2$ and by $\dot{Q}$. In fact, both $\dot{Q}$ and $\dot{V}CO_2$ are finite, and therefore the deviation from line B during CO_2 washout and recovery lies between the extremes described. As the end of a long washout approaches, or as full recovery approaches, a near steady state again ensues, and therefore the deviation from line B diminishes. It can therefore be seen that the net effect of washout and recovery is a loop, the area of which must be inversely related to $\dot{Q}$ and directly related to $\dot{V}CO_2$, i.e.

$$\text{Area of loop} \propto \frac{\dot{V}CO_2}{\dot{Q}} .$$

This experiment is a study of the relationship between $P_{ET}CO_2$ and a rebreathing estimate of $P_{\bar{v}}CO_2$ in two unsteady states beginning from the same control state. The values of $P_{\bar{v}}CO_2$ obtained at the end of each washout at 30 L/min in subject 4 were graphed against the corresponding values of $P_{ET}CO_2$; similarly, values of $P_{\bar{v}}CO_2$ obtained during recovery from 60 min hyperventilation in subject 4 were plotted against the corresponding values of $P_{ET}CO_2$; these are shown in Fig 8. The relationship for all subjects undergoing 20 L/min hyperventilation is shown in Fig 9 and for 30 L/min in Fig 10. The steady state line B from Fig 6 is shown as the dashed line in Figs 8, 9 and 10. It may be seen from Fig 8 that the relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ during hyperventilation may be described as a loop, and that this loop is larger during hyperventilation at 30 L/min than during 20 L/min in Figs 9 and 10. It may also be seen that near the end of washout or near the end of recovery a steady state almost existed and deviation from the steady state line (line B in Fig 6, dashed line in Figs 8, 9 and 10) is minimal.

McEvoy et al (1974) also found, in different circumstances, that the $P_{(v-a)}CO_2$ difference could vary in the unsteady state. They studied patients whose $P_{\bar{v}}CO_2$ ranged from 42 to 84 mm Hg and whose P_aCO_2 ranged from 27 to 65 mm Hg.

It therefore follows that a single estimate of $P_{\bar{v}}CO_2$ may correspond to quite a range of $P_{ET}CO_2$ values. Simultaneous knowledge of $P_{ET}CO_2$

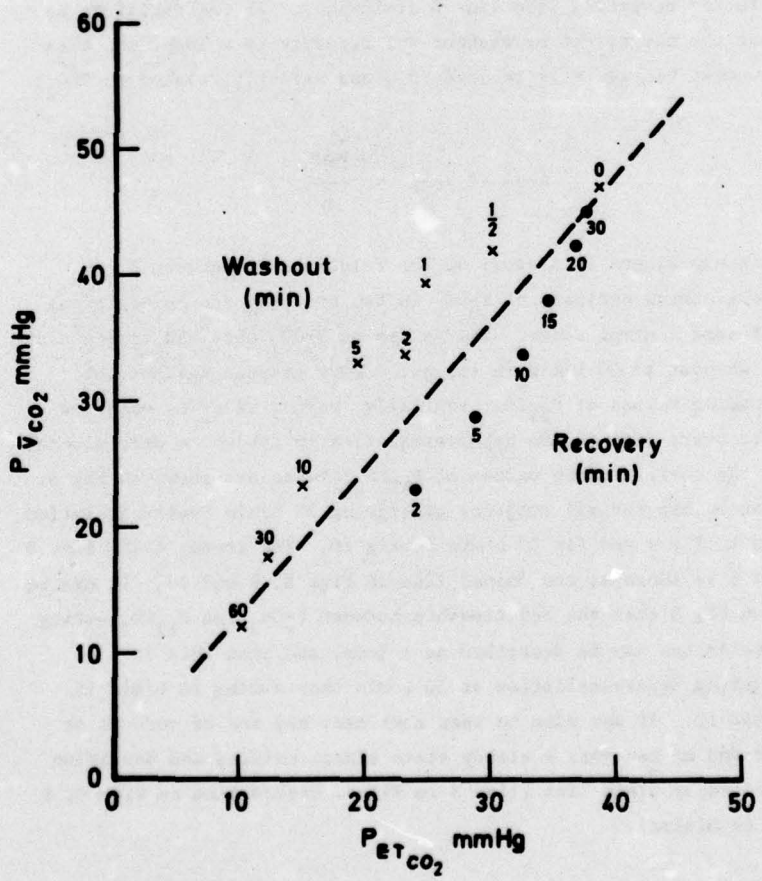

Figure 8. The Time Course of the Relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ during 30 L/min Hyperventilation. (The points *are derived from the End Points of the Washout Periods*, and the Recovery from 60 min hyperventilation, both in subject 4.)

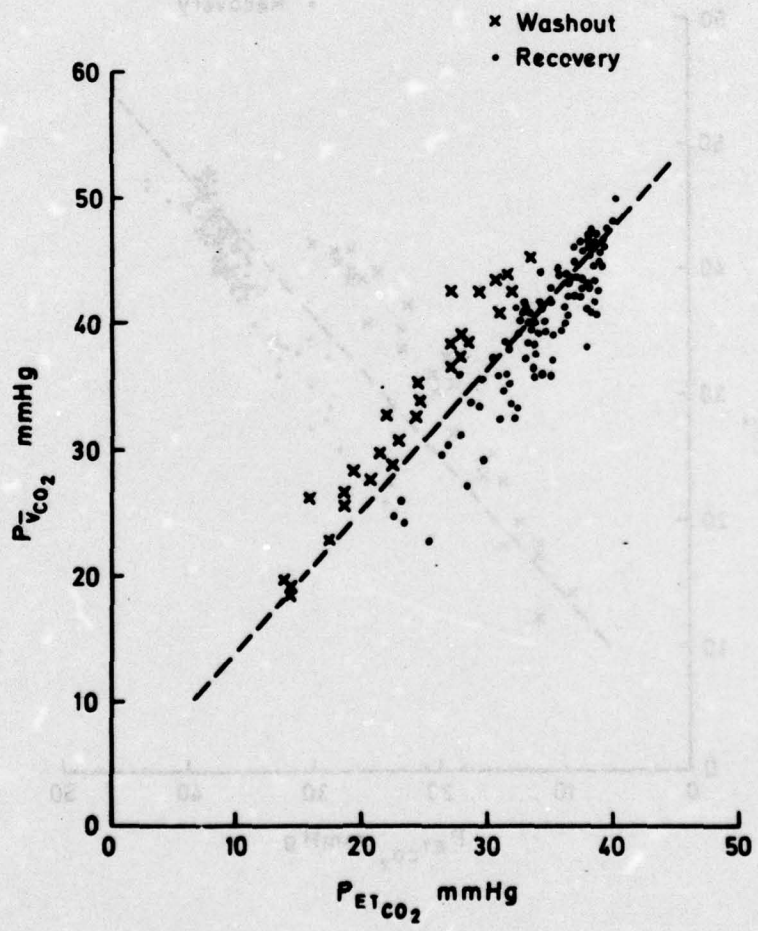

Figure 9. The Relationship between $P_{\bar{v}}CO_2$ and $P_{ET}CO_2$ during 20 L/min Hyperventilation. (Points are derived from the end-point of each experimental washout and the recovery from 60 min washout.)

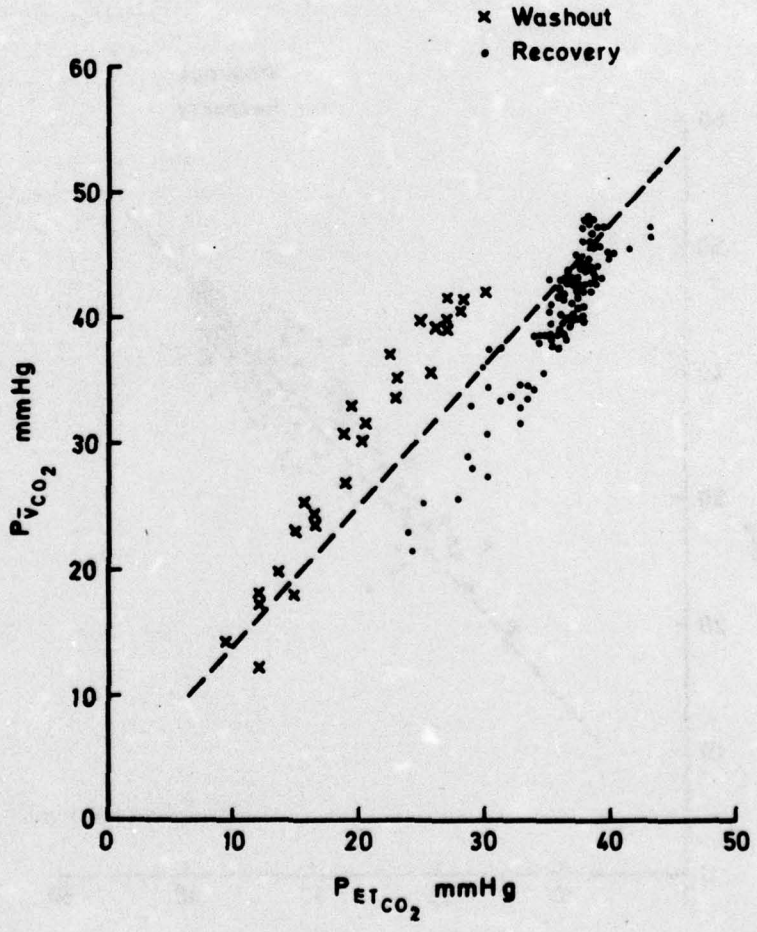

Figure 10. The Relationship between $P_{\bar{V}}CO_2$ and $P_{ET}CO_2$ during 30 L/min Hyperventilation. (Points are derived from the end-point of each experimental washout, and the recovery from 60 L/min washout.)

would only help to fix a point on a loop, but the shape, position of and time course around the loop would be governed by other factors. The implications are therefore that knowledge of the $P_{\bar{v}}CO_2$ value on its own does not help in quantifying previous hyperventilation. This fact, coupled with the exponential nature of recovery, makes the idea of a simple field test for hyperventilation based on a single rebreathing estimate of $P_{\bar{v}}CO_2$ of dubious value.

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